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Course and Prognostic Implications of QT Interval and QT Interval Variability After Primary Coronary Angioplasty in Acute Myocardial Infarction

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OBJECTIVES The aim of this study was to determine the influence of early reperfusion on the course of QT interval and QT interval variability in patients undergoing primary percutaneous transluminal coronary angioplasty (PTCA) in acute myocardial infarction (AMI) and its prognostic implications on major arrhythmic events during one-year follow-up.

BACKGROUND Although early coronary artery recanalization by primary angioplasty is an established therapy in AMI, a substantial number of patients is still threatened by malignant arrhythmias even after early successful reperfusion, which may be caused by an inhomogeneity of ventricular repolarization despite reperfusion.

METHODS Temporal fluctuations of ventricular repolarization were studied prospectively in 97 consecutive patients with a first AMI by measurements of QT interval and QT interval variability during and after successful PTCA (Thrombolysis in Myocardial Infarction flow grades 2 and 3). Continuous beat-to-beat QT interval measurement was performed from 24-h Holter monitoring, which was initiated at admission before PTCA.

RESULTS Reperfusion caused a significant continuous increase of mean RR interval (738 ± 98 to 808.5 ± 121 ms; $p < 0.001$) and a significant decrease of parameters of QT interval (QTc: 440 ± 32 to 416.5 ± 37ms; $p < 0.001$) and QT interval variability (QTcSD: 27.5 ± 3 to 24.9 ± 6 ms; $p < 0.001$) in the majority of patients. However, in patients with major arrhythmic events at the one-year follow-up (sudden cardiac death, ventricular fibrillation or sustained ventricular tachycardia, $n = 15$), parameters of QT interval remained unaltered after successful reperfusion (QTc: 447.3 ± 41 to 432.9 ± 45 ms, p = NS; QTcSD: 35.1 ± 13.4 to 29.0 ± 9.1 ms, p = NS).

CONCLUSIONS Reduction of QT interval and QT interval variability after timely reperfusion of the infarct-related artery may be a previously unreported beneficial mechanism of primary PTCA in AMI, indicating successful reperfusion. (J Am Coll Cardiol 2001;37:44–50) © 2001 by the American College of Cardiology

Cardiac electrical stability may contribute to improved survival in patients undergoing reperfusion therapy in acute myocardial infarction (AMI) (1). Although early reopening of the infarct-related vessel by thombolysis or primary angioplasty in AMI is an established therapy, there are still a substantial number of patients suffering from major arrhythmic events even after successful early recanalization. This fact implies that there may be a persistent injury of myocardium during reperfusion. Sustained electrical transmural injury (persistent ST segment elevations) has been proposed to indicate impaired microcirculatory reperfusion despite successful primary percutaneous transluminal coronary angioplasty (PTCA) (2–5). Ventricular repolarization may also be affected by an impaired microvascular flow, potentially providing a substrate for serious ventricular arrhythmias. Measures of QT interval reflect differences in local myocardial recovery times. Several investigators have suggested that prolonged QT intervals on the 12-lead surface electrocardiogram (ECG) indicate a higher risk for sudden cardiac death after AMI (6–9), independent of age, heart rate and drug use (10). However, there is almost no information available on the course and the prognostic value of beat-to-beat QT interval, a marker of temporal fluctuation of ventricular repolarization, or QT interval variability, a marker of inhomogeneity of ventricular repolarization in reperfused and nonreperfused AMI. Reperfusion may affect QT interval, either directly by influencing the electrophysiological milieu or indirectly by interference with cardiac autonomic nervous control (11).

The aim of this study was: 1) to investigate the influence of early reperfusion on the time course of parameters of QT interval and QT interval variability in patients undergoing primary PTCA, 2) to determine its prognostic implications regarding major arrhythmic events during a one-year follow-up, 3) to examine the possible relationship between abnormalities of ventricular repolarization and quality of reperfusion.

METHODS

Patients. Between August 1997 and June 1998 a total of 116 consecutive patients with AMI admitted to the coronary care unit of the Medical University of Luebeck were

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Abbreviations and Acronyms

ACE = angiotensin-converting enzyme
AMI = acute myocardial infarction
CK = creatine kinase
ECG = electrocardiogram
LDH = lactate dehydrogenase
MAE = major arrhythmic events
PTCA = percutaneous transluminal coronary angioplasty
QTa = QT apex
Te = from the apex of the T wave to its end
TIMI = Thrombolysis In Myocardial Infarction

prospectively enrolled in the study. Inclusion criteria were: presence of ischemic chest pain for more than 30 min but less than 24 h associated with ST segment elevation ≥0.1 mV in at least two leads of the surface ECG. Exclusion criteria were: cardiogenic shock, left bundle branch block, pacemaker rhythm, rhythm other than sinus rhythm, age >75 years, prior AMI or coronary artery bypass grafting and coronary occlusions unsuitable for PTCA. In three patients we did not achieve reperfusion (Thrombolysis In Myocardial Infarction [TIMI] flow grade 0 and 1) (12) by angioplasty. These patients consecutively needed catecholamine treatment and, therefore, were not further followed by Holter monitoring. The final study group was, thus, comprised of 97 patients with TIMI flow grade 2 and 3 and valid Holter recordings. During hospitalization, beta-adrenergic blocking agents and angiotensin-converting enzyme (ACE) inhibitors were given according to evidence-based practice guidelines. All patients gave informed consent for the research protocol that had been approved by the institutional review board.

Catheterization. Coronary angiography and angioplasty were performed by percutaneous femoral approach. Heparin (5,000 to 10,000 U) was administered after arterial access (7F). After visualizing the left and right coronary arteries, left ventricular angiogram was performed in standard projection right anterior oblique 30°. Calculation of ejection fraction was performed off-line by quantitative measurements of end-diastolic and end-systolic area using a computerized algorithm (QLVA, Medis Medical, Leiden, the Netherlands) and was verified by two independent investigators.

Antegrade perfusion of the infarct-related artery was graded according to the classification system of the TIMI trial (12) and was assessed visually. The primary objective was to achieve an optimal angiographic result by PTCA alone. Stent implantation was performed if: 1) TIMI grade flow was <2, 2) residual stenosis was >30% or 3) extensive intimal dissection was present. After the procedure, all patients were admitted to the coronary care unit where they received aspirin 100 mg each per day or additional ticlopidine 250 mg twice a day after stent placement. The femoral sheath was removed after the partial thromboplastin time had normalized. Concomitant medication was given by the decision of the responsible physician. Pain was controlled by intravenous nitroglycerine; however, 18% of the patients received opiates.

Clinical follow-up. After hospital discharge all patients were seen in our arrhythmia outpatients department at 3, 6 and 12 months after AMI. Episodes of nonfatal arrhythmic events were carefully recorded, and information about fatal arrhythmic events and deceased patients was obtained from family members and their general practitioners. Major arrhythmic events (MAE) were assessed after the first 48 h after AMI and were defined as: 1) sudden cardiac death, defined as death within 1 h of the onset of symptoms when detailed case notes were available, 2) survived sudden cardiac death, defined as ventricular fibrillation with consecutive resuscitation and 3) documented sustained ventricular tachycardias. Only the most serious event in the above order was assessed for each patient.

Analysis of QT interval. Beat-to-beat QT interval and time domain QT interval variability were analyzed on a Pathfinder 600 analysis system (Reynolds, Hertford, United Kingdom) from 2-lead 24-h Holter monitoring (Tracker II, Reynolds, Hertford, United Kingdom), which was started at hospital admission. The mean value of each parameter of QT interval and QT interval variability (Table 1) was determined: 1) in the hour before reperfusion, 2) during the first hour of reperfusion and 3) every following hour. Recordings with longer intervals of severe artefacts, imper-

Table 1. Definitions of Parameters of QT Interval Dynamicity and Variability

Variable	Unit	Definition
QT Interval Dynamicity		
QT	ms	QT interval (onset QRS complex to deflection of the T wave to baseline)
QTa	ms	QT apex interval (onset of the QRS complex to the apex of the T wave)
QTc	ms	Bazett corrected QT interval
QTac	ms	Bazett corrected QT apex interval
Te	ms	Interval from apex of the T wave to its end (QT–Qta)
Tec	ms	Bazett corrected interval from apex of the T wave to its end (QTc–QTac)
QT Interval Variability		
QTSD	ms	Standard deviation of QT intervals
QtaSD	ms	Standard deviation of QT apex intervals
QTcSD	ms	Standard deviation of Bazett-corrected QT intervals
QTacSD	ms	Standard deviation of Bazett-corrected QT apex intervals

QTa = QT apex; Te = from the apex of the T wave to its end.

Table 2. Clinical Characteristics for Patients With and Without MAE

	Patients With Valid Recordings (n = 97)		
	no MAE (n = 82)	MAE (n = 15)	p Value
Age (yrs)	59.2 ± 12.5	60.5 ± 11.6	NS
Gender (male/female)	65/17	11/4	NS
Site of infarction (anterior/nonanterior)	40/42	8/7	NS
Left ventricular ejection fraction (%)	55.5 ± 13.3	49.3 ± 15.3	NS
Peak creatine kinase (U/L)	914.9 ± 725.4	1,137.2 ± 544.6	NS
Peak lactate dehydrogenase (U/L)	675.2 ± 402.2	757.9 ± 439.4	NS
TIMI grade (before/after reperfusion)	0.6 ± 0.8/2.8 ± 0.4	0.5 ± 0.7/2.7 ± 0.5	NS
One-vessel disease (n)	33 (40.2%)	5 (33.3%)	NS
Non-Q-wave infarction (n)	8 (9.7%)	1 (6.6%)	NS
Killip class >1 (n)	15 (18.3%)	4 (26.6%)	NS
Diabetes mellitus (n)	15 (18.3%)	2 (13.3%)	NS
Hypertension (n)	47 (57.3%)	9 (60.0%)	NS
Systolic blood pressure on admission (mm Hg)	140.2 ± 24.5	147.5 ± 21.3	NS
Time from pain onset to reperfusion (min)	443.5 ± 267.0	430.0 ± 282.1	NS
Beta-blockers in the acute phase (n)	23 (28.0%)	4 (26.7%)	NS

MAE = major arrhythmic events; NS = not statistically significant.

ceptibility of the T wave, intermittent atrial fibrillation, ventricular fibrillation with consecutive external defibrillation, recording problems caused by excessive sweating and chest movements during the acute phase of infarction and angiographic intervention were not suitable for analysis. A minimum of an 18-h recording time and a minimum of 90% successive QT intervals were required for a tape to be accepted as valid. All tapes were manually edited for exclusion of artefacts and premature beats. To verify the accuracy of the QT measurement, all recordings were reviewed in a large superimposed beat display, indicating the positions of the QT measurement points on each beat. The methods of computerized QT analysis and their reproducibility have been described in detail previously (13–15). To measure QT interval variability we used the standard deviation of all QT intervals per hour. The QT apex (QTa) intervals were measured from the onset of the QRS complex to the apex of the T wave. The T end (Te) intervals (from the apex of the T wave to its end) were calculated using the equation Te = QT − QTa. The Bazett formula was used to obtain heart rate-corrected values of parameters of QT interval and QT interval variability (16).

Statistical analysis. Data in tables are presented as mean values ± standard deviation. Biomedical parameters shown graphically are presented as mean values ± SEM. Statistical analyses were conducted with a commercially available software package (SPSS version 9.0.1; SPSS Inc., Chicago, Illinois). Continuous variables were tested for normal distribution with the Kolmogorov-Smirnov goodness-of-fit test for normality. Discrete variables were compared by chi-square analysis. All measures of QT intervals were normally distributed. Multiple comparisons were done by Bonferroni corrected analysis of variance for repeated measures. Consecutively, an alpha-corrected paired Student *t* test was performed for interval-to-interval comparisons. Spearman's rank correlation procedure was done to determine correlations of parameters of QT interval and QT interval variability with peak cardiac enzymes and left ventricular ejection fraction. Comparisons between groups were performed utilizing a nonparametric independent sample *t* test (Mann-Whitney *U* test). Survival characteristics were analyzed by the Kaplan-Meier method. Statistical significance was set up at $p < 0.05$.

RESULTS

Patients characteristics. Among 116 patients enrolled, a total of 97 patients with a first AMI fulfilled the clinical and technical inclusion criteria. All patients had 24-h Holter recordings with a median duration of 23 h. The clinical characteristics of the patients are shown in Table 2. Successful recanalization of the infarct-related artery was achieved in all patients. During the 12-month follow-up, a total of 19 MAE were documented in 15 patients: sudden cardiac death (n = 4), resuscitation from ventricular fibrillation (n = 7) and sustained ventricular tachycardia (n = 8). Thirteen of these episodes occurred during the in-hospial period (Fig. 1). Subgroup analysis of patients with and without MAE revealed no significant differences regarding age, gender, infarct site, TIMI-flow, left ventricular ejection fraction, Killip class at entry, systolic blood pressure at admission, diabetes mellitus, hypertension and beta-blocker

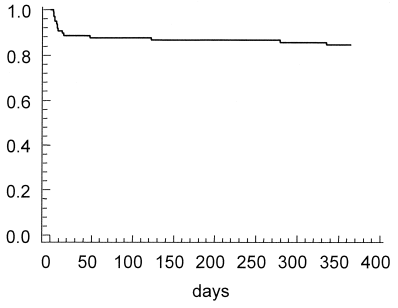

Figure 1. Kaplan-Meier survival curve representing cumulative event-free estimate for major arrhythmic events within a one-year follow-up.

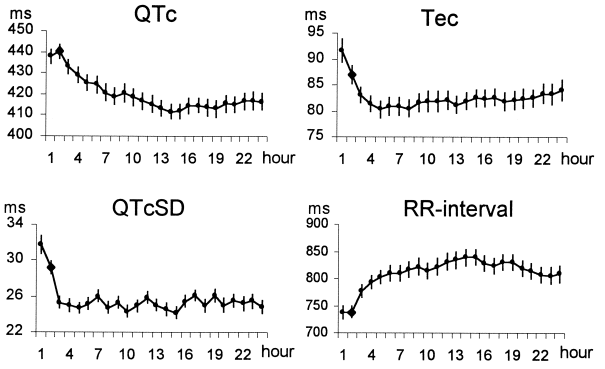

Figure 2. Hourly measurements (mean ± SEM) of QT interval dynamicity (QTc, QTac, Tec), QT interval variability (QTcSD, QtacSD) and mean RR interval during the first 24 h after admission in 97 patients undergoing successful reperfusion by primary percutaneous transluminal coronary angioplasty in acute myocardial infarction. Rhombs = hour of reperfusion.

treatment in the acute phase. Although there were also no significant differences in peak creatine kinase (CK) and lactate dehydrogenase (LDH) levels, patients with MAE tended to have slightly higher values. At hospital discharge and during follow-up, medication did not differ significantly between patients with and without MAE (beta-blockers: 86.7% vs. 91.5%, p = NS; ACE inhibitors: 93.3% vs. 95.1%, p = NS; amiodarone: 6.7% vs. 3.6%, p = NS).

Time course of QT interval and QT interval variability (Fig. 2). After reperfusion RR interval significantly increased from 738.3 ± 97.5 to 776.4 ± 106.4 ms during the first hour after reperfusion ($p < 0.001$), remaining above 800 ms in the subsequent hours. Parameters of QT interval showed a biphasic profile: QTc and QTac moderately increased to their peak levels in the hour of reperfusion (440.2 ± 32.3 ms; 354.1 ± 30.5 ms) and constantly decreased during the subsequent hours, finally reaching levels around 415 ms and 330 ms, respectively, which were significantly above baseline ($p < 0.001$). Without correction for heart rate, the profiles of QT and QTa were comparable; however, they were not as marked as the Bazett corrected parameters, probably due to the significant increase of heart rate after reperfusion. Parameters of the T wave ending (Te, Tec) and parameters of QT interval variability (QTSD, QTaSD, QTcSD, QTacSD) likewise significantly decreased, reaching a stable level between the third and the fourth hour after reperfusion ($p < 0.001$).

QT interval and QT interval variability in patients with major arrhythmic events. In patients with MAE the characteristic biphasic profile of QT interval (QT, QTa, QTc, QTac) was not observed. Moreover, the hourly values were significantly higher after reperfusion than they were for patients without MAE (Fig. 3). There was a marked decrease in parameters of the T wave ending (Te, Tec) after reperfusion in both groups. However, patients with MAE had significantly higher hourly values and, in contrast with the stable decline after reperfusion for patients without MAE, the course of Te and Tec even increased again. There were no significant differences in the hourly profiles of parameters of QT interval variability (QTSD, QTaSD, QTcSD, QTacSD) in either group. Likewise, for patients with and without MAE, a significant decline of these parameters was observed in the first hours, remaining stable in the subsequent hours, with a trend towards higher hourly values in patients with MAE. Furthermore, there were no significant differences in the time course, or in the 24-h mean value of RR interval after reperfusion in patients with and without MAE.

Mean 24-h parameters of QT interval (QT, QTc, QTac, Tec) and QT interval variability (QTcSD and QTacSD) were significantly lower in patients without MAE (Table 3). Although there was a trend towards lower values for mean 24-h QTa, Te, QTSD and QTaSD in patients without MAE, the differences did not reach statistical significance.

QT interval and QT interval variability by perfusion status and infarct site. There were no significant differences in the course or in the 24-h mean value of parameters of QT interval and QT interval variability and mean RR interval among the TIMI flow grade 2 (n = 22) and the TIMI flow grade 3 (n = 75) perfusion groups (QTc 428.6 ± 334 vs. 419.0 ± 30.4 ms; QTcSD 26.3 ± 3.3 vs. 25.6 ± 2.7 ms).

Similar results were observed regarding coronary status and localization of the infarction: parameters of QT interval and QT interval variability did not differ significantly in patients with multivessel (n = 59) versus single-vessel (n = 38) disease (QTc 419.0 ± 27.8 vs. 420.9 ± 33.1 ms; QTcSD 25.4 ± 2.9 vs. 25.8 ± 2.8 ms) and in patients with anterior (n = 48) versus nonanterior (n = 49) infarction (QTc 424.8 ± 29.4 vs. 415.5 ± 32.2 ms; QTcSD 26.3 ± 3.3 vs. 25.6 ± 2.7 ms). Although the difference was not statistically significant, patients with anterior infarction tended to have lower mean RR intervals (782.5 ± 108.2 ms vs. 833.1 ± 109.3 ms).

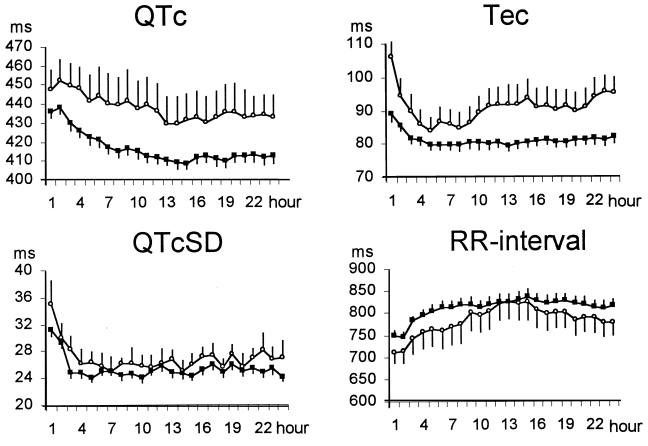

Figure 3. Hourly measurements (mean ± SEM) of QT interval dynamicity (QTc, Tec), QT interval variability (QTcSD) and mean RR interval during successful primary percutaneous transluminal coronary angioplasty in 15 patients with MAE **(circles)** and 82 patients without MAE **(squares)** in a one-year follow-up after AMI. AMI = acute myocardial infarction; MAE = major arrhythmic events; Rhombs = hour of reperfusion.

Table 3. Mean 24-h Values (±SD) for QT Interval, QT Interval Variability and RR Interval in Patients With and Without MAE

	QT Interval					
	QT (ms)	QTa (ms)	Te (ms)	QTc (ms)	QTac (ms)	Tec (ms)
MAE (n = 15)	395.6 ± 53.0	315.0 ± 52.2	80.6 ± 17.3	437.4 ± 46.4	347.6 ± 43.6	89.9 ± 14.8
No MAE (n = 82)	376.2 ± 32.1	303.9 ± 29.6	72.3 ± 10.8	417.0 ± 26.7	336.0 ± 23.4	80.9 ± 13.3
p Value	< 0.05	NS	NS	< 0.01	< 0.05	< 0.01
	QT Interval Variability and RR Interval					
	QTSD (ms)	QTaSD (ms)	QTcSD (ms)	QTacSD (ms)	RR interval (ms)	
MAE (n = 15)	23.3 ± 3.52	20.5 ± 2.8	26.7 ± 2.9	24.1 ± 3.2	795.4 ± 96.9	
No MAE (n = 82)	23.1 ± 2.45	20.4 ± 2.2	25.4 ± 2.8	22.7 ± 2.6	809.2 ± 108.8	
p Value	NS	NS	< 0.05	< 0.05	NS	

MAE = major arrythmic events.

Relation of QT interval, cardiac enzymes and ejection fraction. Significant positive correlations were found between mean 24-h parameters of the T wave ending (Tec) and peak levels of CK ($r = 0.11$, $p < 0.05$) and LDH ($r = 0.26$, $p < 0.05$); however, no significant relations of peak cardiac enzymes and the remaining parameters of QT interval and QT interval variability were observed.

There was a trend for mean 24-h QTc and QTac to correlate negatively with left ventricular ejection fraction ($r = -0.029$ and $r = -0.019$), but statistical significance was not quite achieved. Similar results were obtained when parameters of the T wave ending and QT interval variability were analyzed.

Mean RR interval positively correlated with left ventricular ejection fraction ($r = 0.24$, $p < 0.05$) and negatively correlated with peak CK ($r = -0.18$, $p < 0.05$) and LDH ($r = -0.20$, $p < 0.01$).

DISCUSSION

It is well known that successful reperfusion in AMI reduces subsequent mortality, but it is unclear whether only improved left ventricular function or an increased myocardial electrical stability contribute to this favorable effect (1). By establishing patency of the infarct-related artery, early recanalization may reduce the degree to which an unfavorable electrophysiological local milieu develops.

The principal finding of this study investigating the course of QT interval during and after primary angioplasty in AMI was that early reperfusion resulted in a significant decline of both parameters of QT interval as well as QT interval variability. However, in patients with MAE there was no recovery in parameters of QT interval, even when the infarct-related artery could be reopened.

QT interval and reperfusion in AMI. Although there is growing evidence that abnormal temporal fluctuations in ventricular repolarization are linked to susceptibility to ventricular arrhythmias (17), beat-to-beat oscillations of the QT interval have not been studied in the setting of AMI and reperfusion therapy. The time course of QT interval is a surrogate for these longitudinal variations of the cellular action potential duration, which is influenced by the cardiac autonomic nervous tone as well as the condition of the ventricular myocardial cell itself. In our study patients with MAE a significantly higher 24-h repolarization variability as well as an abnormal course of QT interval after reperfusion was exhibited, indicating repolarization lability and existence of an electrophysiological substrate for severe arrhythmias.

Early reperfusion may reduce ischemia and induce alterations in the electrophysiological characteristics of myocardial cells in the border zone, a critical determinant of arrhythmogenesis after AMI (18). In contrast with previous studies, this study provides direct evidence of significant changes of the course of QT interval with reperfusion in the acute phase of infarction. All parameters of QT interval and their rate-corrected values significantly decreased after reperfusion, indicating a marked decrease of ischemia induced inhomogeneity of the ventricular recovery pattern. As the QT interval is dependent on both ventricular conduction and repolarization, we also investigated the profiles of each component, QTa and Te. It has been proposed that QTa represents the interval from onset of ventricular conduction to the point where 50% of myocardium has repolarized (19) and Te is a pure representative of ventricular repolarization, independent of heart rate and autonomic nervous influence (20,21). The fact that Te and Tec decreased significantly after reperfusion indicates that ventricular repolarization is mainly affected by successful reperfusion. Consistent with our findings, previous studies of patients undergoing PTCA showed significant alterations of ventricular repolarization after reperfusion (22,23).

Autonomic disturbances and impaired cardiovascular reflexes at onset of AMI and after reperfusion of ischemic myocardium have been previously reported (24,25). Successful reperfusion may better maintain the geometry of viable myocardium and thereby prevent activation of sympathetic cardiac autonomic sensory endings that are stimulated by mechanical distortion (26). There is experimental evidence that successful reperfusion is accompanied not only by an increased activation of vagal reflexes, but also by a decrease of reflectoric sympathetic excitation (27). Consistent with these findings, the significant reduction of mean heart rate after reperfusion in our study may reflect a shift of sympathovagal balance towards vagal influence. The fact

that reperfusion in our study caused a significant decrease of both QT interval components as well as mean heart rate suggests a loss of autonomic coupling between heart rate and ventricular repolarization for sympathovagal modulation—a phenomenon that has been described previously in patients with dilated cardiomyopathy or myocardial ischemia (28,29).

Ventricular repolarization and "reperfusion injury." While numerous studies have examined the impairment of microcirculatory flow after successful reperfusion (30–32), data on sustained electrical transmural injury are rare. Recent studies focused on the resolution of ST segment elevation and the extent of T wave inversion in the chest leads on surface ECGs as indicators of microvascular reperfusion injury (4,5,33). However, there is almost no data available on the effects of impaired microcirculatory flow on ventricular repolarization duration and its course after reperfusion. Our study is the first to show an abnormal time course of ventricular repolarization after successful recanalization in those patients suffering from MAE in the follow-up, indicating the maintenance of inhomogeneity of ventricular repolarization and the generation of a possible electrophysiological substrate arising from the impairment of microvascular reperfusion. Furthermore, the positive correlation between peak cardiac enzymes and the duration of the T wave terminus in our study suggests that there is a relationship between the duration of ventricular repolarization and the extent of ventricular myocardial necrosis. As there were no significant differences in ventricular function, coronary status, TIMI flow grade and the remaining clinical parameters, except slightly higher peak values in cardiac enzymes, an absence of recovery of the QT interval may provide independent prognostic information. The reversible impairment of ventricular repolarization after reperfusion for patients without MAE may be interpreted as "electrical stunning" of the ventricular myocardium.

Study limitations. There are several important factors to be considered in interpreting the results of this study. First, continuous measurements of QT interval and QT interval variability are relatively new methods without universally accepted standards of analysis. With the use of our computerized measurement system, however, QT interval was accurately assessed. A second limitation of this study was the fact that many patients received adjunctive drugs such as beta-blockers, benzodiazepines and opiates, with possible influence on ventricular repolarization. However, in our study group, the distribution of patients receiving these drugs was similar in all subgroups, and the drugs did not significantly affect parameters of repolarization. Third, although it is tempting to assume that absence of recovery of parameters of QT interval after successful reperfusion may indicate an impaired microcirculatory flow, no quantitative analysis of coronary flow distribution was performed. Furthermore, no control subjects were investigated in this study. This factor may hamper the interpretation of our findings regarding the effects of coronary recanalization; however, in our study group the total number of patients without successful coronary recanalization (n = 3) was too small for statistical comparisons.

Finally, we would like to point out that it is still premature to classify profiles of QT interval and QT interval variability as indicators of risk after AMI because this study was not designed to test QT interval and QT interval variability as a predictor for ventricular arrhythmias but rather to provide information that would permit design of such a study. The characteristic hourly course of both parameters of QT interval and QT interval variability represents a previously unrecognized phenomenon after reperfusion, which needs further investigation for understanding the underlying cellular mechanisms of repolarization lability in AMI.

Conclusions. Reduction of parameters of QT interval and QT interval variability after timely reopening of the infarct-related artery may be a previously unreported beneficial mechanism of primary angioplasty in AMI. In those patients with major arrhythmic events after successful recanalization, a persistent abnormality of the profile of QT interval after recanalization may be regarded as an electrophysiological correlate of impaired microcirculatory reperfusion. These findings indirectly support the potential clinical importance of temporal repolarization instability after AMI.

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